conservation for function. The direct confirmation that this class II gene is indeed the E_α gene will await gene transfer and expression studies.

We have used a graphically displayed computer routine termed the best-fit matrix analysis (*23*) to analyze possible similarities between the DNA and protein sequences of the E_α gene exons and between these exons and other class II genes, class I genes, immunoglobulin genes, and Thy-1 antigen. Such analyses showed no significant similarities between the different domains of E_α or between the α1 and transmembrane-cytoplasmic domains of E_α and anything other than the same regions of the DR_α cDNA. However, similarity alignment was possible between an area of each of the tested sequences and the α2 domain of E_α. Table 1 lists the sequences compared and the percent homology of the aligned regions to the α2 domain of E_α at both the protein and DNA levels.

The α2 domain of E_α has significant similarity to "homology unit" (*9*) sequences of the genes listed, a sequence associated with the "antibody fold" tertiary structure of antibody domains (Table 1). This observation has been made by several other groups analyzing cDNA (*13, 17*) or genomic (*22*) clones. Of the comparisons made, perhaps the most interesting is that to β_2-microglobulin. Not only is β_2-microglobulin as similar in sequence to any of the class II α2 and β2 domains as these are to each other, but it is strikingly similar in genomic organization to the E_α and DR_α genes. Like these two genes, β_2-microglobulin has its leader peptide and first two codons separated from the main protein coding sequence by a very large intervening sequence (2.8 kb). Of the non–class II exons compared in Table 1, only those of β_2-microglobulin align precisely end to end with those of E_α, employing the same split codon rule. The β_2-microglobulin gene also has the bulk of its 3′ untranslated sequence isolated as a distinct exon some distance 3′ to the last coding sequence (1.1 kb for β_2-microglobulin, 0.8 kb for DR_α).

Though Table 1 suggests that the domains compared diverged from a common anecestor, the evolutionary relationships between the entire genes is unclear. Non-α2-like sequences might have been under much different selective constraints and simply have diverged beyond recognition. It is also conceivable that the α2-like domain has been placed in different genomic contexts through the evolutionary process of exon shuffling (*24–27*). The organizational similarities between E_α and β_2-microglobulin suggest a more direct evolutionary relationship. Regardless, there appear to be fundamental evolutionary relationships among the three classes of genes that regulate and mediate immune responsiveness—Ia antigens, transplantation antigens, and immunoglobulins.

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Ban of DDT and Subsequent Recovery of Reproduction in Bald Eagles

Abstract. *Reproduction of bald eagles in northwestern Ontario declined from 1.26 young per breeding area in 1966 to a low of 0.46 in 1974 and then increased to 1.12 in 1981. Residues of DDE in addled eggs showed a significant inverse relation, confirming the effects of this toxicant on bald eagle reproduction at the population level and the effectiveness of the ban on DDT. The recovery from DDE contamination in bald eagles appears to be occurring much more rapidly than predicted.*

Low rates of reproduction in bald eagle (*Haliaeetus leucocephalus*) populations have caused concern for many years (*1, 2*) and led, in part, to declaring the species endangered. A variety of toxicants, particularly dichlorodiphenyldichloroethylene (DDE), have been implicated in the lowered productivity (*2, 3*). DDE and dichlorodiphenyldichloroethane (DDD) are metabolites of the insecticide dichlorodiphenyltrichloroethane (DDT). Of the three, DDE poses the greatest physiological threat to birds of prey and is the most persistent contaminant in the environment (*4, 5*) and in the bodies of birds (*6*). DDE is so stable in the environment that, in a controlled 11-year study, Beyer and Gish (*4*) were unable to calculate a half-life for the chemical. Evidence of the environmental problems associated with DDT and its metabolites led the Environmental Pro-

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tection Agency to ban further use of DDT effective 31 December 1972 (*7*).

Six years later, Spitzer *et al.* (*8*) reported increased reproduction and decreased DDE contamination in ospreys (*Pandion haliaetus*) in Connecticut and on Long Island. Others have reported scattered improvements in the environment and in bird reproduction (*9*). Similar trends in productivity and DDE levels in addled eggs of bald eagles in northwestern Ontario have now been identified. These eagles nest in a region that has been exposed to little or no DDT, but the eagles migrate in the winter to regions of the United States where they consume DDE-contaminated prey. Reduced DDE contamination was found recently in carcasses and blood plasma of wintering eagles (*10*).

The productivity of the eagle population in northwestern Ontario has been measured annually since 1966. The study area is a region of lakes and boreal forest located between 49° and 53°N and 92° and 95°W. Productivity is calculated on the basis of the number of young raised to the late nestling stage, the last stage at which they reliably can be counted, in all the known breeding areas. A breeding area may be likened to a nesting territory, but the term has fewer behavioral connotations (*3*). An area may contain several alternate nests, but only one nest is used for raising young during any year. The locations and numbers of breeding areas are inferred from proximity of nests and, for uncertain cases, from sequential use over a period of years.

During the study all breeding areas that could be reached each year were counted. Loss of nests, discovery of nests, and logistical constraints influenced the number of breeding areas checked each year. Variation in the number of areas counted is not believed to influence the validity of the productivity estimates because of the relatively large sample size (a minimum of 43 areas, an average of 106) and because comparisons with productivity estimates for nests discovered during random quadrat surveys showed no significant differences. Average annual productivity for 1966 to 1981 is shown in Table 1.

During the 16 years of the study I was able to collect a total of 19 addled eggs. Shell thickness was measured and the eggs were analyzed for several common chemical contaminants. Analyses were performed by the Wisconsin Alumni Research Foundation in 1967 and by the Ontario Research Foundation in the other years (*3*). The results are shown in Table 2.

Statistical comparisons of the productivity and toxicant data are difficult because of small sample size (each year effectively increases N by only 1), considerable year-to-year variation, and the fact that eggs were not sampled at random. Eggs were not collected randomly for logistical, safety, and financial reasons, as well as to avoid disturbing the birds, which might have caused them to abandon their nests, possibly resulting in chilling and lowered survival of embryos. Instead, I waited until after the normal incubation period and collected all eggs that remained. This led to unbalanced sample sizes, and in 8 years no addled eggs were found. In addition, the sample consists of eggs that failed and hence is biased. In spite of these problems, however, I believe that the eggs provide a valid glimpse of contamination levels in the population.

An average level of toxicant contamination for each year in which eggs were obtained was calculated as follows. First I calculated average values for single clutches with more than one egg, on the assumption that eggs in the same clutch are not independent but represent a single female. Then the clutches were averaged. To conduct a regression analysis of productivity on toxicant levels, I used only the 8 years for which both reproduction and toxicant data were available. To adjust for different numbers of clutches during different years, I used the statistical analysis system general linear model procedure weighted for number of clutches.

Productivity in this population of eagles declined irregularly but markedly from 1.26 young per breeding area in 1966 to a low of 0.46 in 1974. Since then it has increased irregularly to 1.12 in 1981, the highest since 1966. In Fig. 1 productivity and DDE, the major toxicant believed responsible for lowered reproduction, are plotted together over the 16-year period. The increase in reproduction following the ban of DDT is highly significant regardless of how the data are viewed. If one considers all 16 years together, for example, the curvilinear, quadratic regression of productivity against year is highly significant ($P < .01$). Alternatively, if the 16 years are broken into two segments represent-

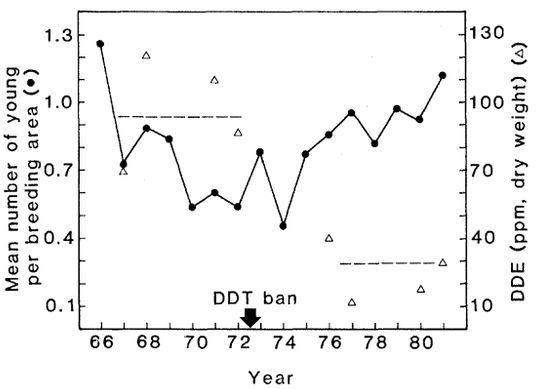

Fig. 1. Summary of average annual bald eagle reproduction and DDE residues in addled eggs in northwestern Ontario, 1966 to 1981. Dashed lines indicate weighted mean concentrations of DDE residues in clutches before (94 ppm) and after (29 ppm) the ban of DDT. Means for the 16-year period are 57 ppm DDE (weighted mean) and 0.82 young per breeding area.

Table 1. Summary of bald eagle reproduction in northwestern Ontario, 1966 to 1981.

Year	Number of breeding areas checked	Number of areas with young	Percentage of areas with young	Number of young	Number of young per nest with young	Number of young per area
1966	43	32	74	54	1.7	1.26
1967	78	41	53	57	1.4	0.73
1968	100	56	56	89	1.6	0.89
1969	106	55	52	89	1.6	0.84
1970	59	21	36	32	1.5	0.54
1971	84	35	42	51	1.4	0.60
1972	94	37	39	51	1.4	0.54
1973	98	44	45	76	1.7	0.78
1974	145	47	32	67	1.4	0.46
1975	118	52	44	91	1.8	0.77
1976	132	71	54	114	1.6	0.86
1977	134	72	54	129	1.8	0.96
1978	117	54	46	96	1.8	0.82
1979	129	75	58	126	1.7	0.98
1980	131	69	53	122	1.8	0.93
1981	129	80	62	144	1.8	1.12
Mean	106	53	50	87	1.6	0.82

Table 2. Organochlorine and mercury residues and eggshell characteristics of addled bald eagle eggs collected in northwestern Ontario, 1966 to 1981.

Year	Nest number	Eggshell thickness (mm)	Ratcliffe index*	Fat (%)	Water (%)	Residues (ppm, dry weight)					
						DDT	DDE	DDD	Dieldrin	PCB's†	Hg
1967	48	NM‡	2.86	3.99	77.9	MI§	44	MI	3.03	NM	NM
1967	71	NM	3.07	8.56	70.3	MI	95	MI	1.21	NM	NM
1968	16	NM	2.71	15.3	71.0	2.62	121	5.17	5.83	NM	NM
1971	48	0.55	2.26	2.4	82.9	0.35	125	13.20	7.37	977	3.51
1971	54	0.48	2.44	2.7	73.8	0.23	95	9.66	5.27	148	2.79
1972	188	0.56	NM	21.8	13.6	ND‖	87	1.86	8.21	176	1.18
1976	325	0.52	NM	5.6	79.0	0.05	13.3	0.76	5.29	64.8	1.00
1976	325	0.53	NM	5.8	78.6	0.05	16.4	0.93	5.42	77.1	0.84
1976	339	0.57	NM	8.8	75.8	0.12	50.4	3.18	12.40	218.2	2.52
1976	297	0.56	NM	6.8	78.6	ND	59.8	1.54	1.31	126.2	1.40
1976	297	0.48	NM	5.8	79.6	ND	56.4	1.91	1.42	121.6	1.57
1977	116	0.55	2.73	2.7	81.9	0.11	0.6	0.61	1.77	91.2	0.44
1977	116	0.53	2.69	5.9	80.6	0.36	23.8	0.82	4.18	186.1	0.41
1980	453	0.58	2.99	4.8	81.1	ND	16.6	1.59	4.02	88.9	1.32
1980	459	0.60	3.20	4.9	77.4	0.09	14.5	0.75	2.70	66.8	4.16
1980	459	0.66	3.16	5.0	78.0	0.09	24.0	1.14	4.09	130.0	3.91
1981	453	0.60	2.97	5.2	81.5	ND	7.8	0.65	1.84	47.5	NM¶
1981	375	0.55	2.63	8.1	79.3	ND	48.2	1.30	1.40	146.4	NM
1981	375	0.60	2.72	8.1	75.1	ND	53.4	1.16	1.41	157.4	NM

*$(M/L \times W)10$, where M = mass in grams, L = length in centimeters, and W = width in centimeters. †Aroclor 1260 was used as the reference standard from 1971 to 1977; Aroclor 1254/1260, believed to be more accurate, was used in the other years. ‡NM, not measured. §MI, measurement inaccurate (interference of PCB's not recognized at the time the measurement was made). ‖None detected (< 0.001 ppm). ¶Mercury was not measured in 1981 becaues the need for this information did not justify the cost of analysis.

ing periods before and after the ban (*11*), linear regression can be performed for the two time periods. The slopes of the two lines are −0.07 and 0.07 young per breeding area per year, respectively. The difference between these slopes is highly significant [t (12) = 3.87, $P < .01$].

For the 8 years for which both reproduction and toxicant data are available, the weighted regression of productivity on DDE is significant ($P < .05$). Levels of DDE in eggs before and after the DDT ban are significantly different ($P < .05$, two-sample rank-sum test).

Other contaminants do not show the same marked relation with productivity. Levels of polychlorinated biphenyls (PCB's) remain relatively high and pose a continuing threat to the eagle population. Levels of dieldrin have not been as high as those of DDE and PCB's, but they are high for dieldrin (*12*). (Dieldrin was banned from further use in 1974.) PCB's and dieldrin may have contributed to or caused the failures of these addled eggs and may have helped suppress overall productivity. Other organochlorines appear to be present only at low levels. Mercury levels were low throughout the study period and not believed to affect the eagles in this region (*13*). Eggshell thickness, measured directly or with the Ratcliffe index (Table 2), generally show the expected relation with high levels of DDE, although sample sizes and variability prevent statistical significance ($P > .05$, Spearman's rank correlation).

These results confirm the suspected negative relation between DDE and bald eagle reproduction at the population level. Although DDE is persistent and may be present in the environment for many years, the effect on bald eagles appears to be diminishing much faster than predicted. It appears that the ban on DDT use was appropriate and effective.

The rapid recovery of eagle reproduction since the ban of DDT is puzzling. DDE contamination in the environment and organisms at the site of original application is known to remain for a long time (*4, 5*). Apparently the toxicant is diffusing out of aquatic food webs, settling into the sediment, or otherwise not continuing to circulate in the bald eagle's food web (*9*).

Even if DDE were to disappear from the prey of these eagles, it would be expected to remain in the breeding birds themselves. Shell thinning and reproductive impairment caused by DDE persist in black ducks (*Anas rubripes*) long after the individuals resume an uncontaminated diet (*6*). DDE, which is fat-soluble, is thought to be eliminated from female birds only through fat deposited in eggs. Once they acquire DDE residues, eagles, which lay many fewer eggs than ducks, would be expected to remain contaminated for long periods, if not for life. If individuals remain contaminated but the population is showing recovery, there may be a higher turnover among breeding birds than is realized. That is, young, relatively uncontaminated females may be replacing older, contaminated birds.

Regardless of the mechanism of recovery, the magnitude of the observed fluctuation in the productivity of these eagles is striking. There are no data on long-term natural variations in the reproductive rates of bald eagles under pristine conditions. I would not expect, however, to see a natural fluctuation in this population on the order that appears to have been caused by the introduction and subsequent withdrawal of DDT.

While the recovery of reproduction in this population is encouraging, it is important to recognize that reproduction is only one dimension of eagle population dynamics. Survival rates and habitat are also important. Changes in survival rates, in fact, may be even more important than comparable changes in reproductive rates (*14*). If a high turnover among breeding birds is responsible for flushing DDE contamination out of the population, it also may signal lower survival rates than expected. We have virtually no good data on survival rates in this species and need to develop techniques to measure survival.

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Asymmetrical Brain Activity Discriminates Between Positive and Negative Affective Stimuli in Human Infants

Abstract. *Ten-month-old infants viewed videotape segments of an actress spontaneously generating a happy or sad facial expression. Brain activity was recorded from the left and right frontal and parietal scalp regions. In two studies, infants showed greater activation of the left frontal than of the right frontal area in response to the happy segments. Parietal asymmetry failed to discriminate between the conditions. Differential lateralization of the hemispheres for affective processes seems to be established by 10 months of age.*

Data derived from both clinical and normal adult samples suggest that certain regions of the two cerebral hemispheres are differentially lateralized for the processing of positive and negative emotional stimuli (*1–7*). This claim is based on (i) neuropsychological findings with brain-damaged patients (*1, 2*); (ii) administration of sodium amytal to patients before neurosurgery (*3*); (iii) the study of lateralized signs and the administration of electroconvulsive therapy to patients with affective disorders (*4*); (iv) studies on normal adults using behavioral indices of asymmetric hemispheric processing (*5*); and (v) electrophysiological studies on normal adults in response to emotional stimuli (*6*). Our view underscores the importance of rostral-caudal differences in hemispheric specialization for affect (*8, 9*). We hold that certain regions of the left hemisphere are specialized for the processing of particular positive affective stimuli while corresponding regions of the right are specialized for the processing of particular negative affective stimuli (*8, 9*). Recent electrophysiological evidence suggests that the locus of this asymmetry is the frontal lobes (*6, 10*).

Although few data are available on the neural substrates of affective development during the first year of life, evidence exists on the development of emotional expression during this period (*11–13*). By 7 to 9 months of age, the infant exhibits a range of both positive and negative affects in response to a variety of specific eliciting situations (*11*). By 7 months of age infants can discriminate among a wide range of facial affects (*12, 13*).

To explore the relation between the ontogeny of affective response systems and cerebral asymmetry, we studied electroencephalographic (EEG) asymmetry in 10-month-old infants in response to positive and negative affective stimuli. By 10 months, infants are capable of both discriminating and expressing positive and negative affect and therefore should exhibit the frontal asymmetry observed in adults. We now report evidence of differential frontal activation asymmetries in response to videotaped presentations of happy and sad facial expressions to 10-month-old infants.

In study 1, 18 healthy female infants ($\bar{X}$ age = 308.1 days, standard deviation = 15.01) born to right-handed parents (*14*) were seen, and artifact-free EEG data on ten subjects were obtained (*15*).

The infant sat in her mother's lap facing a 21-inch diagonal video monitor 45 inches away. The EEG was recorded from the left and right frontal and parietal regions (F3, F4, P3, and P4) with all channels referred to a common vertex (Cz) (*16*) and stored on separate channels of FM tape. Both frontal and parietal regions were recorded because we wished to compare asymmetries in two major cortical association regions (*8, 17*).

The positive and negative affective stimuli consisted of a videotape of an actress generating either a happy or sad facial expression (*18*). Half the infants viewed the happy face first and half the sad face. The audio portion was edited out for both segments.

Artifact-free epochs of EEG were filtered (with cut-offs of 48 dB per octave) for activity within the band between 1 and 12 Hz, integrated, and digitized (*19*). Raw data (in microvolt-seconds) and laterality ratio scores [(R − L)/(R + L), EEG activity (1 to 12 Hz)] were the two dependent measures (*20*).

We first compared happy with sad epochs on the frontal laterality ratio score [(F4 − F3)/(F4 + F3)]. Happy epochs elicited greater relative left frontal activation than sad epochs [$F(1, 9) = 5.88$, $P = .039$] (Table 1). Seven subjects showed higher frontal ratio scores during happy versus sad epochs and two showed equal ratio scores. The parietal ratio score failed to discriminate between epochs [$F(1, 7) = 2.52$].

In order to disentangle the separate contributions of the left and right hemispheres to the frontal asymmetry between the conditions, an analysis of variance was computed on the raw EEG data. A significant condition by hemisphere interaction was obtained [$F(1, 9) = 6.20$, $P = .035$] (Table 2). Happy epochs elicited less activity (1 to 12 Hz) in the left than the right frontal region ($P < .05$, Scheffé test). No difference between the hemispheres in the frontal region was obtained in response to the sad stimuli.

We performed a second study using the identical stimuli and EEG recording and analysis procedure in order (i) to replicate study 1 and (ii) to restrict EEG data analysis to those epochs during

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